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Presentation of Intrapartum Eclampsia with Triad of Posterior Reversible Encephalopathy (pres), Acute Kidney Injury (AKI) and Ascites

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Introduction

Pre-eclampsia complicates 5% of pregnancies globally [1] and the risk of maternal death secondary to pre-eclampsia and eclampsia in developing country is 300 times more than a developed country [2]. PRES is a clinico-neuroradiologic condition, well known to arise in association with eclampsia [3]. It is a neurotoxic state of the brain, usually involving parieto-occipital regions occurring secondary to disruption of blood brain barrier leading to vasogenic edema that usually does not progress beyond posterior cerebral region Jin,2009. Renal injury secondary to hypertensive disorders of pregnancy is another major complication that is frequently observed. Pre-eclampsia is the most common cause of AKI in obstetrics [1].

We report a case of a multipara, known case of pregnancy induced hypertension who presented with intrapartum eclampsia, posterior reversible encephalopathy syndrome (PRES), AKI and ascites.

Case Report

A 40-year-old G4 P30+ at 35+ weeks of gestation, known case of Pregnancy Induced Hypertension (PIH) presented in the Emergency Department with generalized tonic-clonic seizures associated with headache and loss of vision. Her last pregnancy was 7 years back and was non-compliant with medicines.

On examination patient was drowsy (GCS=10/15), her BP was 184/114 mm of Hg and pulse was 111 bpm. On per abdomen examination, uterine size corresponded to 36 weeks. Fetal heart sounds were audible. Per vaginal examination revealed dilated cervix. Immediate management of eclamptic seizures was done. Complete blood count showed 10.4 mg/dL hemoglobin, white blood cell counts (WBCs) of $13.9 \times 10^9/L$. Coagulation profile was normal. Liver enzymes were raised along with uric acid (12.1 mg/dl). Other investigations were BUN 26 mg/dL, Creatinine 1.6 mg/dL, eGFR 35.29 mL/min/1.73 m².

Emergency caesarean section performed due to pathological CTG. Massive ascites of nearly 5 lts were drained from abdominal cavity intra-operatively. Furthermore, whole momentum and peritoneum were found to be grossly studded with nodules, hence, a sample was sent for histopathological evaluation. Alive baby boy with poor Apgar score was born. After a few days, the baby was discharged in stable condition.

Within 4 hours, post procedure she had another single episode of generalized seizure that lasted for 1-2 minutes without any neurologic deficit. MRV and MRA performed that showed patchy abnormal gyral signals in the cortical and subcortical locations of both the parieto-occipital lobes, more marked on the right side [Figure 1]. These appeared

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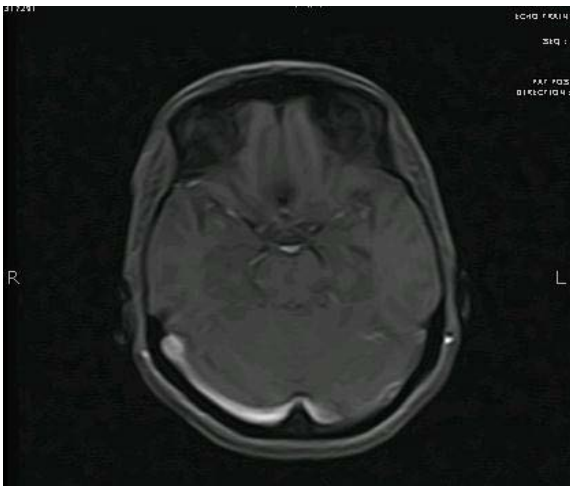


Figure 1: MRI showing hyper intensity in cortical and subcortical area of both sides. Predominantly on right side.

hyper intense on T2/FLAIR sequences. Diagnosis of PRES was made.

A few days later, the histopathological report of the nodular momentum specimen that taken during caesarean section revealed “Metastatic adenocarcinoma”, most likely of the Upper gastro-intestinal tract origin (in the view of immune histochemical profile). This was an incidental finding. She did not have a seizure again during hospital stay and remained stable.

Discussion

PRES was first described as reversible posterior leukoencephalopathy syndrome [4]. Global incidence of this, acute hypertensive encephalopathy. Jin et al. [5] is still unknown [6]. In pre-eclampsia, either high blood pressure disrupts auto regulation in the posterior circulation of the brain, or it is due to endothelial dysfunction causing ischemia, both leads to vasogenic edema of cortical or subcortical regions [5]. Two triggering factors in this case were eclampsia and AKI arising from hypertensive insult contributing to hyper perfusion and edema in the brain. PRES is diagnosed radio graphically in contrast to pre-eclampsia. Management includes aggressive blood pressure control and steps to prevent rise in intracranial pressure as it causes adverse neurological outcomes [6]. It usually resolves over a week if timely treated and MRI changes settle within days to weeks [3]. Complications of PRES include cerebral ischemia, hemorrhage, herniation and death [6].

Second complication observed in this case was AKI. 0.81% of pregnancies experience AKI with the most frequent cause being pre-eclampsia/eclampsia [7]. Placental insufficiency

in pre-eclampsia leads to release of certain mediators from placenta that damages the glomerular endothelium with sub endothelial depositions causing obstruction of capillaries resulting in decrease GFR [7,8]. Acute tubular necrosis in these patients impair tubular secretion of uric acid, leading to hyperuricemia [8] as seen in this case. Treatment of the underlying cause is the cornerstone of management of pregnancy related AKI [8] with a better outcome after treatment [7].

Previously massive ascites of up to few liters have been found during caesarean section in a case. Ascites of nearly 5 lts was drained in this patient and it was believed to arise from capillary leakage due to wide spread endothelial dysfunction secondary to pre-eclampsia [9], but later on histopathology revealed metastatic adenocarcinoma, so ascites in this patient can be due to two etiologies that is pre-eclampsia and metastatic carcinoma. Ascites remain concealed under the false impression of distension due to the fetus.

Conclusion

This case was a series of consequences beginning from gestational hypertension to pre-eclampsia/eclampsia and AKI [3] progressing to PRES. Development of massive ascites can be due to combine result of metastatic adenocarcinoma of upper GI tract and pre-eclampsia. No metastasis identified elsewhere outside the abdomen at the time of presentation in this case.

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